Critical Digest.

ON MULTIPLE SCLEROSIS, WITH ESPECIAL REFERENCE TO ITS CLINICAL SYMPTOMS, ITS ETIOLOGY AND PATHOLOGY.

BY B. SACHS, M.D.

Multiple or disseminated cerebro-spinal sclerosis has for many years been considered to be one of the most easily recognizable diseases of the central nervous system. Under the leadership of Cruveilhier[1] and of Charcot, the French school described the disease so accurately that there appeared to be little to add to the accounts as given by them. The typical forms are indeed unmistakable, but the disease appears so often under all sorts of disguises that its recognition may be by no means easy. Moreover, the interest in it is not exhausted by the establishment of the diagnosis, for it has important bearings to other diseases, and its etiology and pathology present many points for further elucidation. In America the disease is not as frequent as in France and in Germany; at all events, very few contributions have been made to the subject by American writers, and up to the present time I have found but one post-mortem record, and that an incomplete one (by Seguin), published in this country. The time seemed ripe, therefore, for a consideration of the disease; but the present writer will refer only to the more important questions that have been raised regarding it during the last few years, as the work of preceding years has been well summarized in the publications of Charcot, Marie, Gowers, Oppenheim, Dana, Pritchard, and Gray.

---

[1] The bibliography (alphabetically arranged) will be found at the end of this article in the June number.

CLINICAL SYMPTOMS.

The following table of symptoms, as given by Charcot, may be taken as a starting point for the consideration of the symptomatology of multiple sclerosis. It need not be said that all the symptoms are rarely present, and that, according to the predominence of the several series of symptoms, the disease may be divided into a spinal, a cerebral, and a cerebro-spinal type.

I. SPINAL SYMPTOMS.

Positive.	Tremor on voluntary movements of the extremities—"Intention tremor" (arms and head; more rarely of legs). Titubation. Paresis (spasmodic) of the extremities. Contracture, with exaggeration of the reflexes—spastic rigidity.
Negative.	No sensory symptoms, or only very slight disturbance. Vesical disturbance, none or very slight.

II. CEREBRAL SYMPTOMS.

Dysarthria—slowness of speech; scanning of words.
Nystagmus—blank expression.
Attacks of vertigo—spasmodic myosis.
Transitory amblyopia—white atrophy of the papillæ.
Diplopia—associated paralysis of ocular muscles.
Mental enfeeblement.
Apoplectiform and epileptiform attacks.
Difficulty in deglutition.

III. ABNORMAL OR UNUSUAL SYMPTOMS.

Trophic.	Muscular atrophies (amyotrophies), bedsores.
Tabetic.	Lightning pains. Romberg symptom. Anesthetic areas. Vesical and rectal paresis. Gastric crises.

Frequent remission of all the symptoms.

Charcot recognized three distinct periods in the development of the disease:

First period. The disease may begin with cerebral symptoms (attacks of vertigo, transitory diplopia, scanning speech and nystagmus); more frequently the spinal symptoms are the first (a slowly progressive paresis to which intention tremor is soon added); in rare instances there is an apoplectic attack, preceded by vertigo and headaches, and followed by hemiplegia. The rarest of all onsets is the one with gastric crises, which are followed by other more characteristic symptoms. During the second period the typical symptoms are developed, and after years contractures set in. The third period is characterized by mental enfeeblement, speech becomes almost unintelligible, great physical weakness is developed, and finally there are vesical troubles, with subsequent cystitis, bedsores, pyæmic conditions—death. Babinski claimed that an ascending acute myelitis was an occasional cause of death.

Marie has insisted upon a division into four different types, according to the mode of progression: 1. The chronic progressive type—a very gradual increase in all the symptoms and covering a period of years. 2. The chronic type, with exacerbation, marked by the occurrence of hemorrhages, sudden amblyopia, and the like. 3. The chronic remitting type—the progress is very slow, with only slight exacerbations from time to time. 4. The type characterized by permanent improvement and even cure. Almost all of the chronic disorders of the nervous system, tabes, for instance, could be subdivided in the same way. These divisions do not appear to be distinctive; and of the type leading to a complete cure we have seen no instance, and its existence is only a little short of conjectural. To Charcot we owe the recognition of imperfect forms of multiple sclerosis, in which the symptoms that have existed have disappeared. In these abortive forms (formes frustes) some of the important symptoms, as we shall see later on, are never developed, and in others the symptomatology is obscured by the occurrence of hemiplegic, tabic or of amyotrophic symptoms. Oppenheim has at various times directed special attention to the variability of the symptoms, the apoplectiform and vertiginous attacks, and to fundus lesions without functional defect.

The preceding statement is sufficient to show the extreme variability of the clinical pictures as a whole, and explains the possibilities of mistaken diagnosis. During the past few years some of the special symptoms enumerated in Charcot's table have received further elucidation. Struempell, who has a way of upsetting old medical beliefs, has maintained that the intention tremor of multiple sclerosis differs little, if at all, from ordinary ataxia. I have paid close attention to this point during the past year and have at present under observation three undoubted cases of multiple sclerosis, whose movements are distinctly ataxic. On the other hand, it cannot be denied that there are other patients suffering from multiple sclerosis, whose disturbance of motion is more in the nature of a tremor. We believe, too, that a typical tremor is often present at the beginning of the disease, and as the disease progresses the tremor becomes coarser, and then is scarcely to be distinguished from ataxic movements.

Struempell's proposition loses some of its apparent originality if we recall to mind the fact that Gowers in a foot-note (vol. ii., p. 548) states that *ataxic tremor* is the best translation for "Intentions-Zittern." To be sure, he chose the term Ataxic Tremor, "because there is no ataxia without voluntary movements." The difference between ataxia and tremor—a difference in degree only—can be brought out by asking the patient to write with chalk or lead pencil, when it will be found that the ataxic patient scarcely succeeds in the attempt to write, while the patient with tremor writes with some difficulty and in a jerky fashion.

The ocular symptoms of multiple sclerosis have not received the attention they deserve. The other morbid signs had been well understood long before these symptoms were properly described. Our knowledge of them was markedly advanced by the publication of Parinaud, who reported upon the findings in Charcot's cases, and by the monograph of Uhthoff, who described with great detail the clinical and anatomical findings in the eyes of patients suffering from multiple sclerosis. Recent contributions to this special subject have been made by Luebbers—a pupil of Uhthoff, by Guenther, Nagel, Kunn and Schwarz.

The commonest of the eye symptoms are the nystagmus and the nystagmus-like movements. These are most apt to appear on extreme lateral movements of the eyes, and are generally associated in character. While occasional twitching movements of the ocular muscles may occur in healthy persons, regularly developed oscillatory movements upon each lateral excursion are characteristic of the disease. Of course, it should be borne in mind that the symptom is present in other diseases of the nervous system, of which mention will be made later on.

Kunn described a condition which he calls a " fixation tremor" setting in when the eyes are moved from vision straight ahead to the fixation of a definite object. The same author calls attention to a concomitant strabismus which he interprets to be a dissociation of ocular movements that were previously associated.

Luebbers mentions a case in which the nystagmus appeared with converging movements of the eye; but this is entirely exceptional. The nystagmus may be horizontal, vertical, diagonal or rotary. Raehlman and Schwarz dwell upon the special form of ataxic nystagmus, which is practically an uncertainty of movement, the eyes simply overleaping the mark in passing from one point of fixation to another. The same phenomenon is observed in Friedreich's disease, and can be distinguished from the more rapid movements of typical nystagmus. True nystagmus occurs in a majority of cases of multiple sclerosis (according to Uhthoff, in 58 per cent., and according to Marie, in 70 per cent. of all the cases). The variations in the percentages are accounted for by the fact that Uhthoff draws a distinction between nystagmus and nystagmus-like movements. These oscillatory movements of the eyes constitute, therefore, one of the most constant and reliable signs of multiple sclerosis. In view of their frequency, however, it is well to remember that they occur with different forms of congenital defective development (idiocy, hereditary spastic paraplegia, etc.), also in Friedreich's disease, in hereditary cerebellar ataxia, and in a few cases of syringomyelia. Its occurrence in these various diseases is responsible for much of the confusion that has arisen in the attempt to differentiate between them.

Uhthoff is responsible for the statement that affections of the optic nerve are frequently associated with true nystagmus in multiple sclerosis, whereas we are more apt to have nystagmus alone without such atrophy in other conditions.

The actual cause of nystagmus has not yet been fathomed. Uhthoff thought it was due to lesions in the brain, and particularly in the medulla oblongata; but he also concedes that it might be due to changes in the peripheral nerves supplying the ocular muscles. A lesion involving the posterior longitudinal fasciculus was supposed by some to explain the occurrence of nystagmus; but, as Redlich has pointed out, this tract has been entirely exempt in cases of multiple sclerosis with marked nystagmus, and in some cases in which nystagmus was never present, this same bundle was found to be seriously affected. In all probability, any lesion interfering, by lesions of the nuclei or of the associating tracts, with the regular movements of the ocular muscles, would be sufficient to account for nystagmus.

Paralysis of the ocular muscles is not an infrequent accompaniment of multiple sclerosis. It has been present in three of ten cases which have come under my observation within the past two years. The abducens is most commonly affected. Next in order of frequency comes the oculo-motor nerve, which is, however, rarely affected in its entirety. Occasionally the trochlear (IV.) nerve is involved. A point of some importance is that these palsies are variable, disappearnig in some weeks or months, and occasionally returning again to as marked a degree as before. The remission in this one symptom is as characteristic of multiple sclerosis as the remission in most of the other symptoms, and it is the occurrence of such remissions that makes it particularly difficult to distinguish between disseminated sclerosis and multiple cerebro-spinal syphilis (Oppenheim, Sachs).

In addition to single ocular palsies, there may also be paresis or paralysis of associated movements. The upward and downward movements are, as a rule, not affected (Parinaud, Marie, Uhthoff, and Luebbers). Parinaud claimed that paralysis of convergence was frequent, but Uhthoff found this condition in only three per cent. of his cases. A complete ophthalmoplegia externa has not been developed in multiple sclerosis. The direct cause of ocular palsies is to be found in the slerotic patches occupying the region of the various ocular nuclei. Positive and anatomical proof of this would still be welcome.

By way of contrast with the early appearance of ocular palsies in tabes dorsalis, it may be noted that in multiple sclerosis the palsies do not appear, as a rule, until long after unmistakable symptoms of the disease have appeared. The pupils may be unequal, and sometimes react sluggishly to light and during accommodation, but iridoplegia is very rare. Charcot insisted upon the great value of this preserved light reflex as a factor in differential diagnosis. Damtsch claimed that the condition of hippus—a constant changing from contraction to dilatation of the pupil—is often found in multiple sclerosis. He likens the hippus to the increase of the tendon reflexes, but the analogy is not a very happy one.

Much more characteristic of multiple sclerosis than any and all of the preceding ocular symptoms are the changes in the visual power, in the visual fields, and in the structure of the papillæ and the optic nerves. The subjective disturbances are entirely out of proportion (i. e., relatively less), to the objective changes. Complete blindness is the rarest of all occurrences, although transitory blindness has been observed (Schwarz). Complete recovery from the visual defect of multiple sclerosis is possible. According to Uhthoff, Luebbers and others, central scotomata are most frequent. In this area the color sense is the one most disturbed—generally for red and green, sometimes for all colors. These central anesthetic areas may occur together with the normal peripheral fields, or with a mere diminution of peripheral vision. In some cases there may be a peripheral contraction of the visual field with normal central vision. Concentric limitation of the fields has been described by Oppenheim, Thomsen, Buzzard and Uhthoff, but only in cases in which an hysterical or a functional condition was superimposed upon disseminated sclerosis. Almost every writer who has investigated this special subject refers to the disproportion existing between the visual defect and the ophthalmoscopic appearances. In one of Luebbers' cases the visual defect was in keeping with the changes in the papilla, but in two others there was only a very slight atrophy of the papilla, and yet vision was much diminished and the visual fields were distinctly affected. In still another instance there was a marked functional disturbance without ophthalmoscopic change. A few other authors have described distinct changes in the papilæ without any functional disturbance. The visual symptoms generally appear long after the others have been developed, but may come on with extreme suddenness (Uhthoff and Buss).

Gowers and Marie have called attention to the fact that the visual disturbance of multiple sclerosis may be unilateral.

Ophthalmoscopic examination has revealed several peculiar changes. Complete atrophy of the optic nerves is very rare. Uhthoff found it in only 3 of 100 cases. The rarity of this is worthy of note, and is probably due to the frequent preservation of the axis cylinders, in spite of the destruction of the medullary sheath. Incomplete atrophy

—discoloration of the entire papilla—the peripheral parts being more distinctly altered than the nasal half, occurs in about 50 per cent. of all cases. Atrophy of the temporal halves of the papillæ is almost pathognomonic of multiple sclerosis. Charcot insisted that the papilla retained a yellowish color in contradistinction to the white appearance in the atrophy of tabes. Optic neuritis has been found in a number of cases (in three of Luebbers' cases). Partial temporary atrophy is, according to Uhthoff, a common symptom in intoxication of amblyopias, tobacco and alcohol); but in these the atrophy is always bilateral, whereas it is frequently unilateral in multiple sclerosis. This special resemblance to the intoxication amblyopias is of interest, if we remember that multiple sclerosis has been shown to be at times of toxic origin.

The anatomical examination of the eyes (Uhthoff, Luebbers, and others) has revealed conditions as variable as are the functional disturbances. It has been shown that there are widespread atrophic changes in the optic nerves, depending probably upon the proliferating process emanating from the connective tissue, and gradually involving the nerve fibres; the sheaths are often destroyed, but the axis cylinder is, as a rule, not affected; in other words, the process is interstitial in origin, the changes in the nerves being secondary. In consequence of this preservation of the axis cylinders, the secondary degeneration does not ensue, and the nerve fibres do not forfeit their functional power.

It is very evident, as will be shown in the section on pathology, that the sclerotic changes in the optic nerves are very much the same as in the other parts of the central nervous system. The next question is, whether or not the changes in the connective tissue are due to the disease of the blood-vessels. No unanimity of opinion has as yet been reached on this point, and it is more than probable that the process begins differently, according to the varying etiology of the cases. Uhthoff, Buss, Taylor and others have paid especial attention to the behavior of the blood-vessels. Several of these writers have found an increase in the number of the finer blood-vessels, and a change in their walls, followed by a proliferating process in the surrounding tissue.

Nolda, who studied the relations of multiple sclerosis to infectious diseases, thinks that the sclerotic patches take their origin from the walls in the blood-vessels. Luebbers, in comparing the changes in multiple sclerosis with those found in the primary optic atrophy of tabes dorsalis, finds that in multiple sclerosis there is more of a focal degeneration than in tabes, and, above all, there is in tabes no such number of normal axis cylinders as in multiple sclerosis. The optic atrophy of tabes is due to the primary degeneration of the optic nerve fibres, while in multiple sclerosis the atrophy is the result of an interstitial neuritis, and this in turn may be due to a disease in the blood-vessels. In multiple sclerosis, it may be added, the retinal elements are not affected.

Scanning speech has long been considered a characteristic symptom of multiple sclerosis; but it would be a mistake to suppose that this is the only speech disturbance common to this disease. Putting it broadly, almost every form of dysarthria is possible in multiple sclerosis, for the disturbance is due to lesions in the pons or medulla interfering with the speech mechanism. Leube found a disturbance of innervation of the vocal cords in one instance, and I have recently seen a typical case of the disease in which the opposition of the vocal cords was so imperfect that the patient spoke in whispers. In other cases nasal speech has been noted, and in still others true bulbar speech has been reported. In the writer's own experience, there is often an evident speech tremor not unlike that observed in the early stages of general paresis, and reminding one somewhat, too, of the peculiarities of speech in some patients suffering from Friedreich's disease. Disturbances of articulation, if excessive, are apt to be associated with other symptoms pointing to an involvement of the various nerve nuclei in the pons and the medulla oblongata (cases of Joffroy, Leyden, Berlin, Fuerstner, Redlich and others). If these symptoms predominate, they constitute the special bulbar form of multiple sclerosis. This type was carefully considered by Hallopeau.

An interesting example of this class of cases was recently reported with autopsy by Schuster and Bielchowsky. In this instance nasal speech was associated with paresis of the left arm and the left leg, with dyspnœa, headaches and double vision. There was also divergent strabismus of the right eye, as well as paralysis of both sixth nerves. The pupils and fundi were normal. A focal lesion in the pons was suspected, but the autopsy revealed multiple sclerotic foci in the pons and medulla and in other parts of the brain.

Aside from the peculiarities in the development of single symptoms, abnormalities occur in the development of the entire clinical picture of multiple sclerosis. In the literature of the subject the formes frustes—the aborted types of multiple sclerosis—have given rise to considerable discussion. It is well to bear in mind that there is no regularity in the development of sclerotic patches, whence it follows that departures from the typical forms of the disease will occur readily enough. Slightly irregular forms do not, therefore, deserve the designation "formes frustes."

Charcot was the first to dilate upon these special types, referring particularly, 1. to cases in which certain symptoms, which had existed, had disappeared; 2. to cases of which the symptomatology had never been complete—such cardinal symptoms as the tremor or the nystagmus never having developed; 3. to cases in which the type has been altered by the intervention of other symptoms of an hemiplegic, tabic or amyotrophic order.

The establishment of the formes frustes has opened the door to diagnostic errors; for a multiple sclerosis may appear under the guise of an hemiplegia, of a transverse myelitis, or of an amyotrophic lateral sclerosis. In the opinion of the present writer, the diagnosis of multiple sclerosis should be made only if the cardinal symptoms of the disease, such as nystagmus, scanning speech, intention tremor are present in addition to the other symptoms of myelitis, hemiplegia, etc. The variability of the symptoms (Oppenheim) argues in favor of multiple sclerosis, as do also the recurrent apoplectic and vertiginous seizures. Oppenheim has laid especial stress upon the occurrence in such cases of the characteristic changes in the fundus, with little or no functional impairment of vision.

Apoplectic attacks may occur in the course of a multiple sclerosis, but occasionally, as in a case now under my observation, hemiplegia is developed slowly at the outset of the disease, and is then scarcely to be differentiated from other forms of apoplexy; excepting that in these cases which affect men and women in middle life the usual etiological factors of an apoplectic seizure, cardiac disease, or specific endarteritis are generally absent. If the person who has suffered from an apoplectic attack develops marked nystagmus, scanning speech or ataxic movement in the paralysis of the upper extremity, the suspicion of multiple sclerosis may well be entertained. Cases of this character have been described by Werner, Bikeles and others. The question arises whether these apoplectic attacks in multiple sclerosis are due to vascular disturbances or to a peculiar localization of the sclerotic patches. I am inclined to side with Charcot, Redlich and Babinski, who believe that these forms are due to the early development of sclerotic patches in the pons and medulla oblongata, although Taylor has shown that such patches also occur in the cortex and other parts of the brain. Witzel has published a case in which there was a crossed paralysis of the facial and abducens nerve of the opposite side.

Many writers have conceded the occurrence of acute and chronic cases of multiple sclerosis, presenting none but spinal symptoms (Bourneville, Guérard, Leyden and Oppenheim have recorded cases of acute, transverse myelitis which were eventually proven to be cases of multiple sclerosis). In the absence of the ordinary etiological factors (transverse myelitis trauma, syphilis, vertebral disease) the suspicion of disseminated sclerosis may be entertained, but the cardinal symptoms of the disease must be in evidence to prove the suspicion.

The purely spinal and chronic forms may appear under the clinical guise of a spastic spinal paralysis. For months, and even years, no other symptoms may be forthcoming. No doubt, some of the cases of primary lateral sclerosis were in reality early forms of multiple sclerosis. Cases of this description have been reported by Krueger, Ormerod, Lapinski and Redlich. A very typical case of this character was observed recently in my department at the Polyclinic, to which it had been referred by Dr. A. Wiener. In all such cases the occurrence of the charcteristic changes in the fundus will lend valuable support to the diagnosis of multiple sclerosis. However clearly the symptoms may be developed, and however carefully, multiple sclerosis may be confounded with other diseases, above all with cerebral spinal syphilis (Oppenheim, Sachs and others). The remissions in the symptoms, the preponderance of the spasticity over the paralysis, the apoplectic seizures may be characteristic of both diseases. Greater stress, in my opinion, should be laid upon the pupillary changes in syphilitic affections (absolute immobility, unilateral pupillary symptoms and irregular contour of the pupils), and the presence in multiple sclerosis of nystagmus, intention tremor and scanning speech, which are, after all, extremely rare in syphilis of the brain and of the spinal cord.

There will be, as a rule, no difficulty in differentiating between multiple sclerosis and paralysis agitans; but Schultze and Sachs have reported upon cases in which the symptoms of multiple sclerosis have been associated with those of paralysis agitans; in one young patient of the latter writer, who exhibits most of the symptoms of paralysis agitans, there are also a nystagmus and marked nasal speech. In another patient, whose condition was at first diagnosticated (by competent observers) as functional tremor, then as intention tremor of multiple sclerosis, later developments have shown the disease to be a typical form of paralysis agitans. In view of such experiences, we may maintain that the clinical distinction between multiple sclerosis and paralysis agitans is not as absolute as it has been supposed to be since the days of Charcot.

We may concede that hysteria may simulate multiple sclerosis. More often hysterical symptoms are present in addition to those of multiple sclerosis. The differentiation will depend largely upon the presence of such distinctly hysterical stigmata as are foreign to the pure type of multiple sclerosis. Westphal's two cases, in which the anatomical changes were wanting, although the patients exhibited all the symptoms of the disease, have led to much discussion.

French writers have called them hysterical without much hesitation, and Struempell has chosen to establish a separate form of pseudo-sclerosis, bringing under this head what appears to us a rather heterogeneous number of cases. He has allied with this form a diffuse sclerosis of children; but the clinical types which he attempts to establish need careful examination before they can be adopted.

This leads to the consideration of multiple sclerosis in early life. The differential diagnosis between it and other diseases of infancy presents so many difficulties, that much care should be exercised in making the diagnosis, or in accepting it when offered by others. In a recent article by Struempell, in which the author challenges criticism on other points, he insists that the diagnosis of multiple sclerosis in children is not certain, unless corroborated by post-mortem findings, and to this conservative view we are inclined to subscribe.

The first case of multiple sclerosis in a child appears to have been described by Schuele (1871). This was followed by reports of Dreschfeld, Bristowe and Ten Cate Hoedemaker. Marie, in 1883, reported upon cases of his own, and collected 13 in all. In 1887 Unger had increased this list to 19. Six years later, Totzke, reporting 2 cases of his own, tabulated 39 in all. One case was reported by the present writer in his book on the "Nervous Diseases of Children," which Stieglitz has, through a printer's error, called a doubtful case in the latest publication on this subject. Stieglitz cites three cases of his own, and tabulates 35, evidently excluding some which he considered doubtful. In addition to those diseases of the adult which enter into the discussion of the differential diagnosis, such as syphilis, transverse myelitis, hysteria, we must especially consider the infantile spastic palsies, hereditary spinal ataxy (Friedreich's disease), hereditary cerebellar ataxy and hereditary spastic paralysis—the last has been omitted from Stieglitz's carefully prepared list.

As for the infantile cerebral palsies, the ordinary paraplegias (birth cases and cases of defective development) will generally be ruled out by the history of early onset, by the frequent occurrence of convulsions and by defective development, which is more marked than in cases of multiple sclerosis.

The acute hemiplegias, with their characteristic symptoms, cannot give rise to any confusion. Some doubt could be entertained, however, with respect to the cerebral diplegias. Nystagmus is common enough in these; intention tremor, or, rather, ataxic movements, are observed frequently, and disturbances of speech (bradylalia) are very common. It is very rare, on the other hand, to find any two of these symptoms in a case of diplegia. Furthermore, it should be remembered that a cerebral diplegia of an acute onset is a rare occurrence, and rarest of all in patients beyond the age of five to seven years. These cerebral diplegias present extreme contractures and post-paralytic disturbances of motion (athetosis and the like), which seldom occur in multiple sclerosis. As a matter of fact, of the large number of cases of cerebral diplegia which the present writer has seen, there has not in a single instance been reason to suspect the presence of multiple sclerosis. In making the diagnosis, the entire congeries of the symptoms should be considered, and not any one clinical sign. Stieglitz says, correctly, that the question is whether infantile cerebral palsy has been excluded in cases diagnosticated as multiple sclerosis.

From Friedreich's disease differentiation is generally easily made, although every now and then patients come under one's observation whose clinical symptoms may leave some doubt as to the presence of one or the other of these diseases. A resemblance may be due to the peculiarities of speech, to the awkward movements of the upper extremities in Friedreich's disease, which may simulate an intention tremor,and to the presence of nystagmus; but the condition of the reflexes in disseminated sclerosis, the optic atrophy and the absence of true ataxia of the lower extremities will help to establish the differential diagnosis; and, after all, in Friedreich's disease the disturbances of speech are slow and labored, but rarely of the typical scanning order.

At a recent meeting of the Neurological Society I presented an adult patient in whom the symptoms pointed to Friedreich's ataxia, and yet there was a bare possibility of his having a bulbar form of multiple sclerosis. This patient was 39 years of age, his past history was good, without any evidence of preceding syphilis. At the age of 20 he was weak in the knees, and frequently made missteps. In 1887 he had sought medical advice because of difficulty of locomotion, noticed especially in climbing stairs. When examined in March, 1895, he complained chiefly, while walking, of weakness in the extremities and slight difficulty in speech. He exhibited an ataxic spastic gait and static ataxia. The pupils reacted to light and during accommodation. The patellar reflexes were absent. There was distinct ataxia of the right upper extremity. He had a form of speech which was midway between a slow and a bulbar speech. There was slight atrophy of the optic nerves. The case was at first supposed to be one of locomotor ataxia, but the further development of the disease showed that it was either Friedreich's ataxia or a bulbar form of multiple sclerosis. The intention tremor, the optic atrophy and the peculiarities of speech would be in harmony with the diagnosis of multiple sclerosis; but the absence of the knee-jerks, ataxia of the upper extremities, which was not a typical intention tremor, and the normal behavior of the pupillary reflex were in keeping with Friedreich's ataxia, which appears, however, as a rule, early in life. The first symptoms in this patient's case appeared at least as early as the age of 20 years, and possibly earlier.

Still greater difficulty, however, is experienced in the attempt to differentiate multiple sclerosis from hereditary cerebellar ataxia (type, Nonne-Marie). This is well illustrated by Stieglitz's Case II., in which the diagnosis of multiple sclerosis hangs by a hair, and the author himself does not appear to be firmly convinced of the diagnosis. If a previous history of ptosis, of a paresis of the internal rectus muscle and the Romberg symptom do not support the diagnosis of hereditary cerebellar ataxia, it is also certain that swaying of the body, with the eyes open and closed, is not very usual in multiple sclerosis. In a case like this, as well as in the one cited above by me, the post-mortem examination alone will satisfactorily determine the nature of the disease. It is well to insist again upon the fact that the mode of onset, the time of life at which the disease is developed, as well as the history of some preceding illness, will often help us to differentiate between the two diseases.

Hereditary, spastic paralysis, as described by Newmark, Pelizaeus and others, should be considered in this connection. There is a striking resemblance in the symptoms, but nystagmus, intention tremor and scanning speech are not typical of the hereditary form of spastic paralysis. Only a few months ago a boy, 12 years of age, came under my observation, who, after a fright, had developed marked spastic paralysis of the lower extremities, with slight ataxic movements of the right upper extremity, and a peculiarity of speech, which was slow and deliberate, rather than scanning. There were no nystagmus, no pupillary abnormalities, no optic atrophy—in short, no symptoms that were at all characteristic of multiple sclerosis, except the speech. In this case the fact that the boy was absolutely well until the age of 12, that there was no history of any similar occurrence in any member of his family, and that the disease came on in rather abrupt fashion, argued largely in favor of multiple sclerosis; and yet, but for the slight ataxic movements of the right upper extremity and the peculiarities of speech, there was little to throw the balance in favor of disseminated sclerosis. In cases of this description time is needed for the development of further symptoms, which will enable one to establish a diagnosis with some degree of certainty.

(*To be concluded in the June number.*)